Evidence for a Black–White Crossover in All-Cause and Coronary Heart Disease Mortality in an Older Population: The North Carolina EPESE

ABSTRACT

Objectives. This cohort study evaluated racial differences in mortality among Blacks and Whites 65 years and older.

Methods. A total of 4136 men and women (1875 Whites and 2261 Blacks) living in North Carolina were interviewed in 1986 and followed up for mortality until 1994. Hazard ratios (HRs) for all-cause and cause-specific mortality were calculated, with adjustment for sociodemographic and coronary heart disease (CHD) risk factors.

Results. Black persons had higher mortality rates than Whites at young-old age (65–80 years) but had significantly lower mortality rates after age 80. Black persons age 80 or older had a significantly lower risk of all-cause mortality (HR of Blacks vs Whites, 0.75; 95% confidence interval [CI] = 0.62, 0.90) and of CHD mortality (HR 0.44; 95% CI = 0.30, 0.66). These differences were not observed for other causes of death.

Conclusions. Racial differences in mortality are modified by age. This mortality crossover could be attributed to selective survival of the healthiest oldest Blacks or to other biomedical factors affecting longevity after age 80. Because the crossover was observed for CHD deaths only, age overreporting by Black older persons seems an unlikely explanation of the mortality differences. (*Am J Public Health.* 1999;89:308–314)

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Racial differences in mortality have been widely documented in the United States.[1] According to US vital statistics data, Black Americans have higher mortality rates and shorter life expectancy at virtually all ages from birth onward.[2] Among middle-aged and older adults, cardiovascular disease mortality is the most important determinant of the disadvantage that Blacks experience.[3] By around age 80 years, however, all-cause and cardiovascular disease mortality rates among Black men and women decline to lower levels than among Whites,[3–5] a phenomenon referred to as mortality crossover.[6–9]

Much of the evidence for the mortality crossover comes from vital statistics data, and the validity of the crossover has been challenged because of the potential for racial differences in age misreporting by decedents before death or family members after death.[10] Previous studies of 2 separate cohorts of Black and White men[5] indicated that older Black men tend to have lower mortality than older White men. Longitudinal epidemiologic studies in biracial populations of men and women can provide important data for determining whether the mortality crossover actually exists and, if so, whether it may be explained by specific risk factors for mortality.

The North Carolina site of the Established Populations for Epidemiologic Studies of the Elderly (EPESE) study was funded by the National Institute on Aging to examine racial differences in health and mortality in older persons. This unique database, one of the largest observational studies involving Black and White persons 65 years and older, makes it possible to assess how race affects all-cause and cause-specific mortality in an older biracial population. The mortality crossover can be evaluated in a population in which age was ascertained as many as 8 years before death. Furthermore, the extensive data collected in the study allow examination of whether racial differentials in mortality change when the risk estimates are adjusted for demographic and health-related risk factors, such as differences in income, lifestyle, and health status.

Methods

For this study, we used data from the 5-county North Carolina community of the EPESE, a multicenter longitudinal study of persons 65 years and older. A 4-stage stratified household design was used to construct a probability sample of persons 65 years and older in Durham, Franklin, Granville, Vance, and Warren counties. The sample was designed to contain at least 50% Black persons, although older Blacks represented only 35% of the population. Blacks were oversampled to facilitate examination of racial differences. Details of the method have been published elsewhere.[9,11] Briefly, between 1986 and 1987, trained interviewers

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conducted household surveys to collect baseline information from 4162 people (80% of eligible respondents), of whom 2261 were Blacks and 1875 were Whites. Participants (n = 26) who were not classifiable as Black or White were excluded from these analyses. Participants were followed up annually for 6 years with in-person and telephone interviews until 1993.

Variable Classification

Sociodemographic variables included each respondent's sex, age, race, urban or rural residence, use of Medicaid, years of education, and annual personal income. Participants were asked both their age and their date of birth, and for any inconsistencies the date of birth was used to calculate current age. Personal income information was not available (refused or did not know) in 18.5% of the population. Income was therefore categorized in 4 groups: <$5000/year, $5000–$9999/year, $10 000/year (considered as the reference), and a missing income group. Smoking history was coded as never, past, or current smoking, and body mass index (BMI) was computed as weight in kilograms divided by height in meters squared from self-reported data. BMI information was missing for 11% of the participants. BMI was classified in 5 groups: <20 kg/m^2, 20–25 kg/m^2 (considered as the reference), 25–30 kg/m^2, >30 kg/m^2, and missing BMI.

Health status variables included self-reported history of heart attack, stroke, diabetes, treated hypertension, presence of mobility, and physical disability related to activities of daily living.[12] The presence of angina and intermittent claudication symptoms was assessed with the Rose questionnaire. Use of selected drugs, considered as indicators of disease presence and severity, included nitrates, diuretics, and digitalis. Blood pressure, measured with the Hypertension Detection and Follow-Up Program protocol,[13] was the mean of 2 readings obtained while the subject was seated. Systolic or diastolic blood pressure measurement was not available for 6.5% of the participants. Blood pressure levels were categorized in 4 groups: diastolic blood pressure <90 mm Hg and systolic blood pressure <140 mm Hg (normal blood pressure, considered as the reference); diastolic blood pressure <90 mm Hg and systolic blood pressure 140–160 mm Hg (borderline systolic hypertension); diastolic blood pressure <90 mm Hg and systolic blood pressure ≥160 mm Hg (systolic hypertension); and diastolic blood pressure ≥90 mm Hg and any systolic blood pressure (diastolic hypertension). The measurements missing for blood pressure, income, and BMI were coded as separate dummy variables. All other covariates included in the models had less than 3% missing data: subjects with missing data for these covariates were all pooled in the reference categories.

Mortality Follow-Up

Information on vital status from 1986 through December 1994 came from annual follow-up contacts and linkage with the National Death Index and was virtually complete (>99%). The death certificate was obtained and coded by a single nosologist by using the *International Classification of Diseases, Ninth Revision* (*ICD-9*).[14] Death due to coronary heart disease (CHD) was defined as *ICD-9* codes 410–414 as the underlying cause of death. Death due to other cardiovascular diseases (not including CHD) was defined as *ICD-9* codes 401–409, 415–459, and 250 as causes of death. All other deaths were pooled in one category and were considered as noncirculatory deaths.

Statistical Analyses

Weighted prevalence estimates of demographic and health-related characteristics were computed on the basis of the calculated weights, which take into account race, sex, age, location of residence, and nonresponse to the study. The weights allow for projection of the sample data to reflect the status of the same aged population (approximately 28 000) in the study's geographic area.

Smoothed, age-specific all-cause and CHD mortality hazard curves, with age as the time variable and stratified according to race, were computed using Breslow's estimate[15] with a program written in S-PLUS.[16] The Breslow's estimate is naturally suited to the present study's staggered entry; that is, persons enter the risk set at their own age at study entry and not at time zero of the study. The hazard curves presented are the unweighted Breslow estimates that have been smoothed using locally weighted regression.[17]

Crude death rates were obtained by dividing the number of events by the accumulated number of person-years. This was done separately for age groups 65 to 70 years, 71 to 79 years, and older than 80 years, with person-years and events partitioned into the appropriate age groups. Estimates of the Black-to-White hazard ratios were calculated for these 3 age groups and were derived from unweighted Cox proportional hazards regression models computed with the PHREG procedure in the statistical package SAS Version 6.12.[18] This technique allows age-specific hazard ratios to be estimated by using age and not follow-up time as the time variable. All subjects were entered in the analysis at the time of their age at entry in the study (age at baseline) and exited the cohort at the age at their death or their censoring and were considered at risk for the event during this period. To account for the transition through age groups (from 65–70 to 71–79 or from 71–79 to 80+) during the follow-up time, 3 sets of proportional hazards models were computed. The first set of models included only participants who were aged 65 to 70 years at baseline. Of these, those who survived beyond 70 years were censored at that age. The second set of models included participants who had survived beyond 70 years and persons who were aged 71 to 79 years at baseline. Of these, persons who survived beyond 80 years were censored at that age. The last model included participants who had survived beyond 80 years and subjects who were 80 years and older at baseline. For those who shifted from one age group to the older age group, follow-up time was partitioned according to the time spent in each age group.

Multivariate models were run with and without subjects with missing data to assess the effect of removing those with missing information. Risk estimates were in the same direction and of similar magnitude, although confidence limits were wider because the sample size was reduced. Final analyses were run with 3 separate dummy variables for missing blood pressure, income, and BMI, whereas subjects with missing data for other variables were pooled in the reference categories. As shown in previous research,[19] when less than 5% of the data are missing, there is very little need to do multiple imputations of missing data. In this data set, it has been demonstrated that including these subjects in the reference category results in a very small amount of bias, even if bias is present.[19]

To assess the effect of the complex survey design on the results, Cox proportional hazards regression analyses were performed with PROC SURVIVAL in SUDAAN[20] to incorporate the sample weights, the stratification, and the clustering in the analysis. The results were consistent with minimal effects of the complex survey design on the main conclusions derived from unweighted estimates. Therefore, the estimates from the unweighted Cox regression models are presented. Weighted percentages are shown in Table 1 to provide prevalence rates of the risk factors that are generalizable to the population from which the sample was selected.

Results

Table 1 presents weighted age-specific prevalence estimates of demographic characteristics, CHD risk factors, and chronic conditions according to race.

TABLE 1—Weighted Prevalence Rates (%) of Select Demographic Characteristics, Coronary Heart Disease Risk Factors, and Chronic Conditions by Age Groups and Race: Duke EPESE, 1986–1987

	65–70 Years		71–79 Years		80+ Years	
	White (n = 728)	Black (n = 921)	White (n = 784)	Black (n = 881)	White (n = 363)	Black (n = 459)
Female	58.1	59.7	63.0	63.9	70.1	66.5
Income $10 000/year	53.4	19.2	39.0	12.6	21.3	6.2
Education <9 years	26.3	55.8	31.6	68.4	42.3	73.1
Use of Medicaid	2.6	9.5	2.5	9.4	4.9	12.5
Isolated systolic hypertension	6.4	7.1	12.6	10.3	15.3	10.8
Diastolic hypertension	15.4	25.8	13.4	18.8	9.2	14.3
BMI >30 kg/m^2	14.8	26.7	9.7	23.3	7.1	9.9
Former smoker	36.9	26.0	31.6	25.3	19.7	17.6
Current smoker	23.6	24.4	16.5	13.6	5.6	5.2
Diabetes	11.7	23.1	13.6	22.1	8.5	17.6
Heart attack	12.4	9.9	14.2	11.2	14.6	11.2
Treated hypertension	33.8	47.0	38.0	48.3	32.3	38.0
Use of diuretic and digitalis	4.5	4.1	5.9	4.9	10.9	9.8
Stroke	6.6	8.5	5.2	8.9	10.9	7.0
Mobility disability	20.2	26.0	28.5	35.3	54.0	61.5
ADL disability	6.2	7.8	8.7	11.2	20.6	19.8

Note. EPESE = Established Populations for Epidemiologic Studies of the Elderly; BMI = body mass index; ADL = activities of daily living.

During 21 618 person-years of follow-up, 1284 people died, 289 from CHD (*ICD-9*: 410–414), 293 from other circulatory diseases (*ICD-9*: 401–409, 415–459, 250), and 702 from all other causes. The smoothed age-specific mortality hazard curves, using age as the time variable and stratifying according to race, are shown in Figures 1 and 2 for all-cause and CHD mortality, respectively. Between 65 and 75 years of age, Black mortality tends to be higher than White mortality. From 70 to 75 years of age, White mortality rates rise more quickly than Black rates, resulting in a convergence of the 2 hazard curves and a mortality crossover between 75 and 80 years of age. Blacks who survived until 80 years of age showed an age-associated acceleration in mortality after 80 years of age that was not as steep as the acceleration observed among Whites. This results in a divergence of the 2 survival curves through very old age.

This crossover pattern suggests that different hazards related to race are present for all-cause and CHD mortality over time (with age considered as the time variable). When hazard ratios are not constant over the time variable, the proportionality assumption is violated and a race-by-age interaction can be postulated. The presence of this interaction was formally tested by including in a proportional hazards model the main effects of race and age and a race-by-age interaction term, which was significant for CHD mortality ($P = .04$).

To further describe this mortality crossover, and to account for this age-by-race interaction, an age stratification was performed in age groups 65 to 70, 71 to 79, and older than 80 years, and a race term was included in the analyses. Age-specific rates were also analyzed by sex, in order to disclose sex-specific effects in determining racial differences. Crude age- and sex-specific death rates for all-cause and CHD deaths are presented in Figure 3 according to race. Before 80 years of age, all-cause and CHD mortality rates in Blacks were higher than rates in Whites. After 80 years of age, all-cause and CHD mortality rates were markedly lower in Blacks than in Whites. The mortality crossover was more marked in men but was present in both sexes. The presence of a race-by-sex interaction was formally tested in multivariate models, and because the interaction term was not significant, further analyses were performed in men and women combined.

To model the mortality crossover, separate age-specific proportional hazards models were run for CHD, other cardiovascular diseases, other-cause, and all-cause mortality. The Black-to-White hazard ratio by age groups and for all-cause and cause-specific mortality are presented in Figure 4. All models were adjusted for CHD risk factors, demographics, and health-related variables. When we compared fully adjusted mortality risks, no significant differences in mortality risks between Blacks and Whites were found before 80 years of age (Figure 4). After 80, Blacks had a decreased risk for all-cause and CHD mortality. After adjustment for potential confounders, the Black-to-White hazard ratio was 0.75 (95% confidence interval [CI] = 0.62, 0.90) for all-cause mortality and 0.44 (95% CI = 0.30, 0.66) for CHD mortality (Figure 4). The Black-to-White hazard ratio for other cardiovascular disease mortality and other causes of death were not significantly different from 1 (Figure 4).

Among those 80 years and older, the relation of Black race to all-cause and CHD mortality was not altered by adjustment for demographic characteristics, CHD risk factors, and presence of chronic conditions. As shown in Figure 5, the stepwise inclusion of groups of adjusting variables did not remove racial differences and minimally affected the hazard ratio estimates: from an unadjusted hazard ratio of 0.81 (95% CI = 0.69, 0.95) to an adjusted hazard ratio of 0.75 (95% CI = 0.62, 0.90) for all-cause mortality and from an unadjusted hazard ratio of 0.59 (95% CI = 0.42, 0.83) to an adjusted hazard ratio of 0.44 (95% CI = 0.30, 0.66) for CHD mortality.

Discussion

The results of this longitudinal study of a biracial older population confirmed previous observations from vital statistics data that a mortality crossover between Blacks and Whites occurs around 80 years of age; after this age, Black older persons survive longer than White older persons. This study also yielded important information for the interpretation of this demographic phenomenon: first, in cause-specific analyses, the mortality crossover was observed for CHD deaths only (Figure 4); second, when demographic and health-related risk factors are taken into account, racial differences in mortality persisted and lower mortality rates for CHD and all-cause mortality were still observed in Blacks after age 80 (Figure 5).

This study adds important clues for further interpreting and understanding racial differences in mortality.[21,22] One strength of this

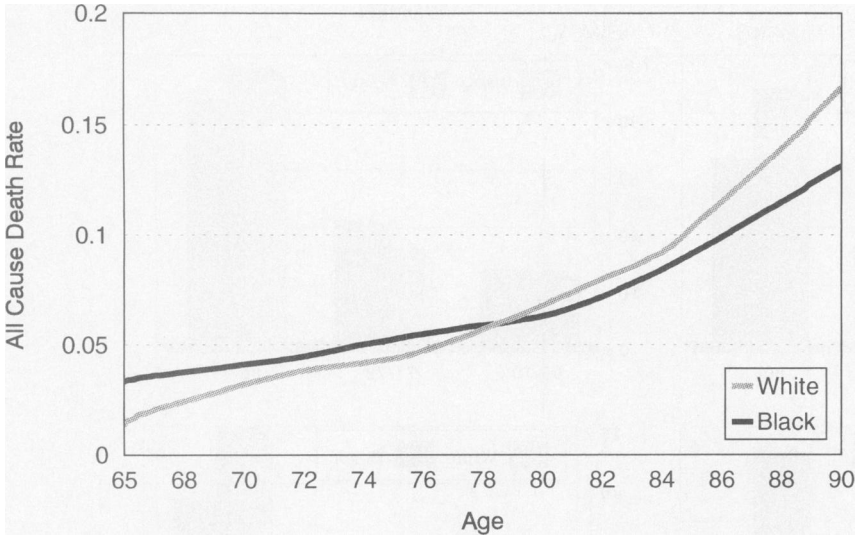
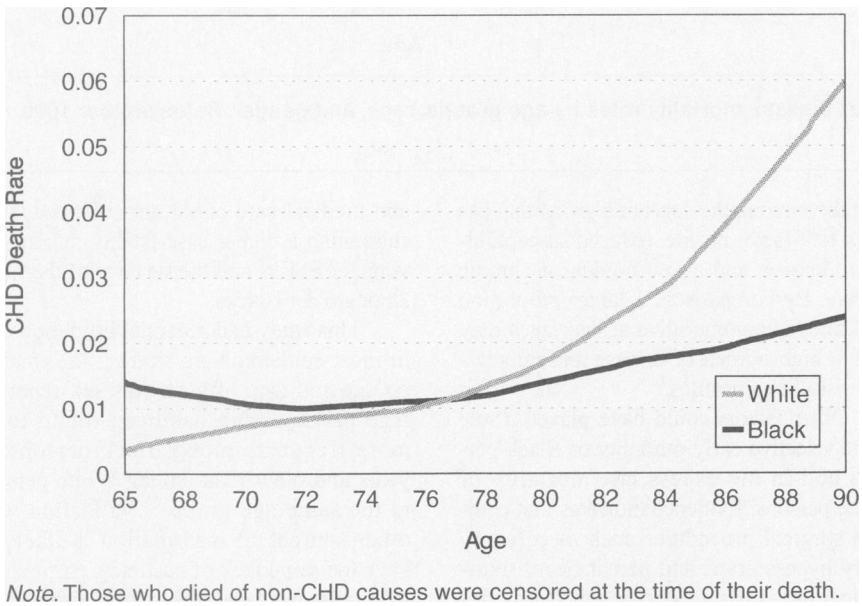


FIGURE 1—Smoothed age-specific hazard curves for all-cause mortality, according to race.



Note. Those who died of non-CHD causes were censored at the time of their death.

FIGURE 2—Smoothed age-specific hazard curves for coronary heart disease (CHD) mortality, according to race.

study is in the use of cause-specific mortality and in the inclusion of several health-related risk factors in the multivariate analyses. This allowed us to test several hypotheses for explaining the racial differences in mortality and to model the mortality crossover after removing the effects of potential confounders of the relation between race and mortality. A second strength of the study is in the racial composition of the study cohort. This is the ideal setting for studying racial differences, given the high proportion of Black participants and the diversity in demographic characteristics of both the White and the Black study participants. Finally, most study participants were women, and the observation of a mortality crossover in this cohort expands on previously observed mortality crossovers in male cohorts.[5]

Age overreporting and systematic misreporting among Black elderly have been hypothesized as possible explanations for the mortality crossover observed in vital statistics data collected from death certificates.[10,23,24] Most of these studies have found inaccuracies in age reporting when age at death on death certificates was matched with other sources of information, such as birth certificates, previous census data, Social Security records, and self-reports of age by decedents years before death. In one study,[10] the mortality crossover disappeared after the corrected age for Blacks was used, although age for Whites in this study was self-reported and not subject to the intensive age validation done for Blacks.

In our longitudinal study, age and date of birth were recorded in the same manner for Black and White participants (and in a small proportion, from proxies) up to 8 years before death, so participants' ages were more likely to be accurate than ages on death certificates (as those ages were reported by relatives or other sources that are less reliable than the participants themselves).[25] Furthermore, we hypothesized that if age misreporting is the explanation for the mortality crossover, the mortality crossover should be observed across all causes of death. For age overreporting to explain a mortality crossover for a single cause of death in a prospective study, differential age overreporting would have to be related to the disease an individual would eventually die from, which is very unlikely. In our study, the mortality crossover was observed only for CHD mortality and not for other deaths, suggesting that the mortality crossover is a real phenomenon and is not merely the result of age misclassification.

Two possible explanations for the mortality crossover should be considered. The first postulates a cohort effect. Those born before 1910 might have lived through periods when certain lifestyles and risk factors were more favorable for Blacks and allowed this cohort to live longer than Whites, once they reached old age. This effect could be hypothesized to result from factors such as high-fat diet, sedentary life, and smoking's being less prevalent among Blacks. This lower prevalence was found to be the case in the 1960s,[26,27] the period when this cohort was approximately 40 years and older. If this hypothesis is true, this mortality crossover should disappear in the next decades, when Blacks from later cohorts with less healthy lifestyles will be reaching old age.[28]

Cohort effects in the Black population older than 65 may have resulted in much greater heterogeneity than in the older White population. Heterogeneity for health outcomes in the Black populations has recently been demonstrated according to birthplace and migration. Fang et al.[29] categorized Black Americans in New York City according to whether they were born in the

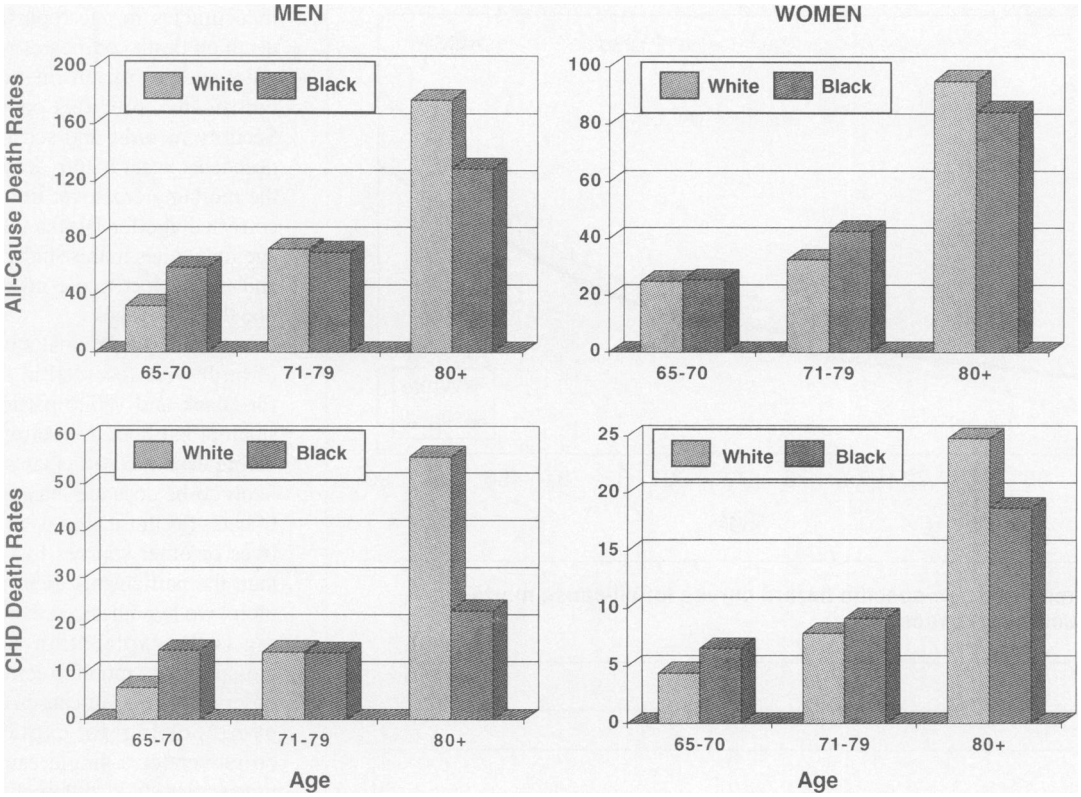


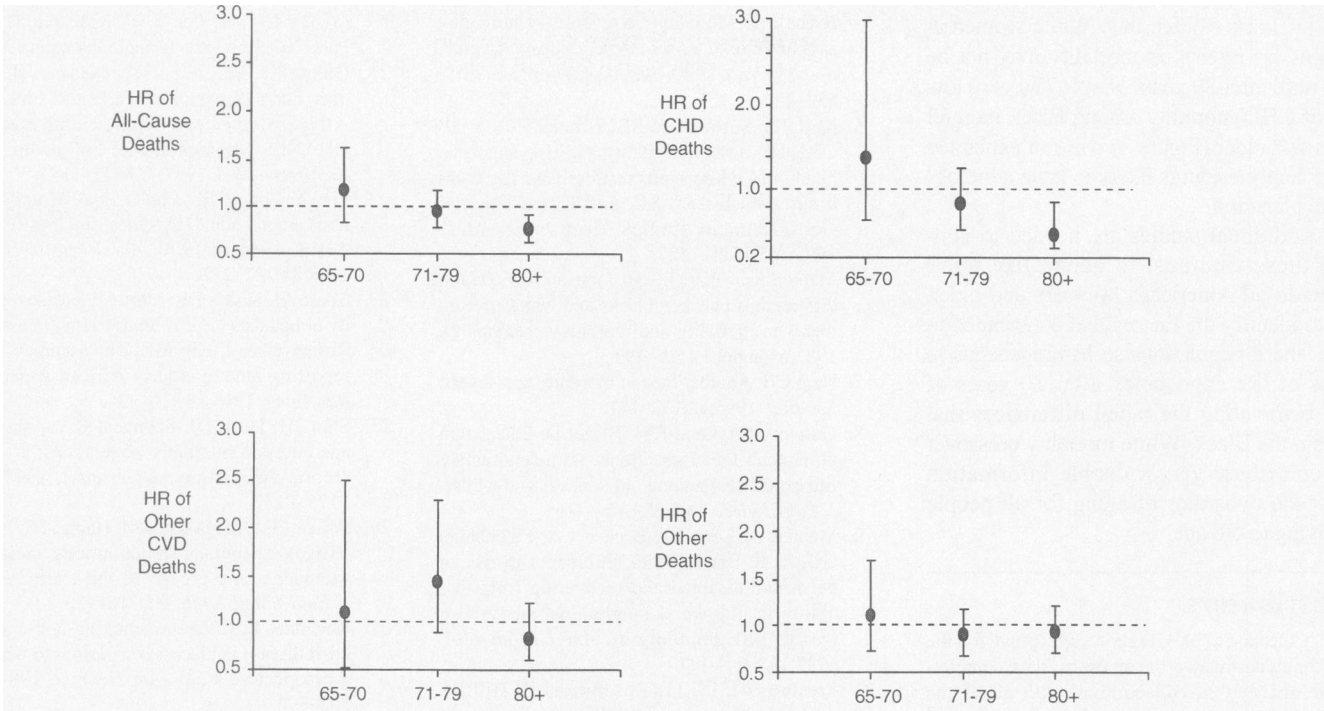
FIGURE 3—Crude all-cause and coronary heart disease mortality rates by age groups, race, and gender. Rates are per 1000 person-years.

southern United States, the northeastern United States, or the Caribbean. Blacks born in the South had the highest mortality rates from cardiovascular disease, those born in the Northeast had intermediate mortality rates, and those born in the Caribbean had the lowest mortality rates. As suggested by Gillum,[30] a dynamic interpretation of demographic and social transitions in the Black population since the last century explains its heterogeneity. Differences in acculturation, urbanization, and lifestyle that he postulated are responsible for dramatic changes in cardiovascular disease may be important distinguishing features of the young-old and old-old Blacks that make up our study population. Although survival certainly affects the age trends in socioeconomic, behavioral, and health-related characteristics seen in Table 1, there may be some indication that younger Blacks are becoming more educated and more affluent but also have acquired harmful habits such as smoking and have a higher prevalence of overweight, hypertension, and diabetes.

The second line of explanation for the crossover includes the selective mortality of high-risk Blacks at younger ages. A bimodal distribution or at least extreme heterogeneity of risk-factor exposure in the Black population could lead to early death in Blacks who have a high risk-factor profile but to low mortality among the surviving group that has a low risk-factor profile, reduced susceptibility to disease, and a low burden of chronic disease. By comparison, a larger proportion of Whites remaining alive at later ages may have a high burden of disease and unfavorable risk-factor profiles.[6,31]

Other factors could have played a role in the selective early mortality of Black persons and in the excess late mortality of White persons. Evidence indicates that common surgical procedures such as coronary artery bypass grafts and percutaneous transluminal angioplasty are performed less frequently among Black than among White Medicare beneficiaries.[32] Additional evidence also exists for racial disparities that are unfavorable for Blacks in procedures such as mammographies, limb amputations, and influenza immunization.[32] Among men screened for the Multiple Risk Factors Intervention Trial, income, as a measure of socioeconomic status, has been shown to be the most important determinant in Black–White all-cause mortality differences; however, for cause-specific mortality, the role of other sociocultural and biological factors could not be investigated.[33] Given the higher prevalence of chronic conditions such as heart attacks and stroke among older Whites in our study, less access to preventive and medical care could also be postulated, suggesting a higher case-fatality rate among younger Blacks and the survival of the fittest among older Blacks.

This study had potential limitations. As in most epidemiologic studies, information on age and date of birth was self-reported. Age misreporting has been found to be more frequent among Black persons 80 years and older than among White persons in the same age group.[24] Validation with other sources of information is likely to have the same lack of accuracy, particularly among Blacks born before 1915, when birth certificate registries were mandated for all states. For these subjects, therefore, only indirect evidence can be used to confirm whether the mortality crossover is a true demographic phenomenon or an artifact of age misreporting. Another limitation is that lipid levels were not available at baseline, and racial differences in high-density lipoprotein and total cholesterol levels could not be accounted for. Racial differences in lipid levels have been documented, particularly in high-density lipoprotein cholesterol levels, which are more favorable among older Blacks than among older Whites.[34] These differences, however, have been described for the entire population 65 years and older and therefore are an

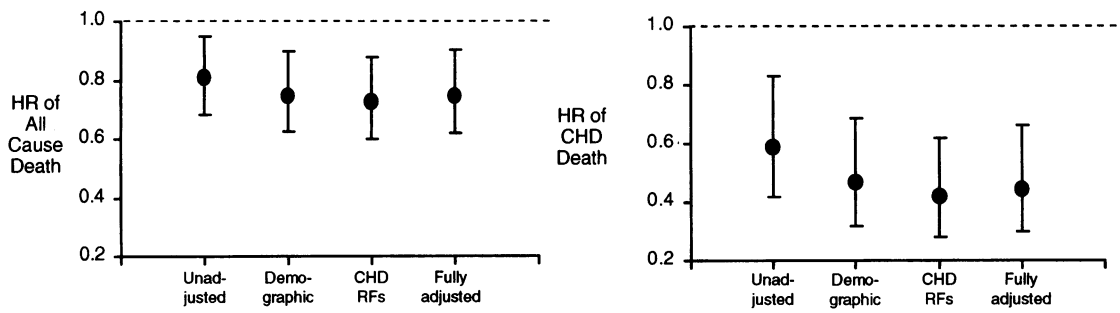


Note. From 12 separate proportional hazards models adjusted for race, personal income, education, urban/rural residence, use of Medicaid, body mass index, measured blood pressure, smoking, history of diabetes, stroke and heart attack, treated hypertension, and positive Rose questionnaire for angina and claudication. Other conditions added in the fully adjusted models were use of selected drugs (digitalis, diuretics, and nitroderivatives), mobility, and disability related to activities of daily living. Above 1, Whites have a higher mortality risk; below 1, Blacks have a lower mortality risk.

FIGURE 4—Black-to-White hazard ratios comparing Black race vs White race by age group and cause of death.

unlikely explanation of the differential survival probability observed by age group.

In our study, the rate ratios remained practically unchanged even after adjustment for health status and other coronary risk factors, suggesting that other, still unmeasured, complex factors contribute to this epidemiology of excess mortality among Whites older than 80 years or exceptional longevity among very old Blacks. We could control for urban vs rural residence, but this would not take into account other potentially important consequences of the cohort effect, such as stages of urbanization, acculturation, and lifestyle transitions. Furthermore, biological and genetic differences may have a greater impact at late ages and may be a plausible explanation for the mortality crossover. For



Note. The unadjusted models included only the Black/White race term (Black vs White). Demographic variables included personal income, education, urban/rural residence, and use of Medicaid. Coronary heart disease risk factors included body mass index, measured blood pressure, smoking, history of diabetes, stroke and heart attack, treated hypertension, and positive Rose questionnaire for angina and claudication. Other conditions added in the fully adjusted models were use of selected drugs (digitalis, diuretics, and nitroderivatives), mobility, and disability related to activities of daily living. Above 1, Whites have a higher mortality risk; below 1, Blacks have a lower mortality risk.

FIGURE 5—Black-to-White hazard ratios in the subgroup 80 years and older with stepwise cumulative inclusion of adjusting variables from 8 separate proportional hazards models.

example, although osteoporosis is less common in Black women than White women at all ages, its effects on mortality may not be seen until after 80 years of age. The very low rate of CHD mortality among Black men 80 years and older (Figure 3) was not explained in our analyses but is likely to have a biological explanation.

Additional studies are needed to confirm these findings, to generalize these results to all Americans 80 years and older, and to identify the factors that contributed to select these populations so heterogeneous in terms of life expectancy after 80 years of age. Unraveling the racial differences that explain the Black–White mortality crossover can contribute very valuable information about the dynamics of aging for all people surviving to old age. □

Contributors

M.-C. Corti and J. M. Guralnik conceptualized the study and coordinated the analyses; they completed the analyses in collaboration with all of the authors, particularly M. Pahor, S. G. Leveille, and L. Ferrucci, experts in the field of cardiovascular and geriatric epidemiology. R. J. Havlik, J. M. Guralnik, H. J. Cohen, and C. Pieper were involved in the study planning, study sampling, and data collection and collaborated in the interpretation of the analyses. G. Izmirlian and L. Ferrucci actively collaborated in the statistical analyses and designed several of the figures.

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